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Editorial: Changing views on myopia

In 1604 Kepler proved that in myopia parallel rays of light come to a focus in front of the retina and it was assumed that the reason for this state of refraction lay in the length of the eyeball. Such a theory was particularly attractive in the nineteenth century when the climate of philosophical opinion was summed up by Lord Kelvin who said that he could understand nothing of which he could not make a model. Arlt in 1856 examined enucleated myopic eyes and found grounds for the acceptance of this theory. Although further measurements of excised globes suggested that other factors might play a part in the aetiology, the idea of axial myopia had so taken hold of the scientific imagination that investigators failed to consider other possibilities. The ophthalmoscope, invented in the 1850s, revealed temporal crescents at the optic disc and retinal changes affecting the posterior pole of the myopic eye. These seemed to support Kepler's theory because their clinical appearance was that of stretching of the choroid.

Once this mechanism was generally accepted it was natural to inquire why it was that some globes grew larger than others. It was believed that the eyeball could be lengthened mechanically by contraction of the extraocular muscles. Excessive accommodation with overacting medial rectus muscles were inculpated so atropine was given to paralyse the ciliary muscle and tenotomy of the horizontal rectus was practised. Some considered the oblique muscles with their relatively posterior position as the more likely compressors of the globe and they also were attacked surgically. Others thought that the globe might be lengthened because of a rising intraocular pressure, so that eserine had its supporters and some surgeons even performed iridectomy to keep the tension low. Because myopia was known to appear and progress in young scholars, school hygiene received vigorous attention. Special schools were built and run with emphasis on the minimum of close work, good lighting, large print, adequate diet, and work in the open. Myopia was thus regarded as a pathological condition calling for therapy, either medical or surgical.

As a result of this effort myopia has certainly not disappeared, nor has its progress been limited. A move forward in the understanding of this state of refraction depended upon a fresh point of view. In 1913 Steiger made this step by recognizing that the conception of axial myopia could not explain all cases and, in his search for a further variant, he found that corneal refraction ranged from 38 to 48 D. He postulated, after careful investigation, that corneal refraction and axial length were freely variable components determined genetically, and that their chance union could produce any refractive error. His theory broke the stifling obsession that axial length is the whole cause of myopia, it brought attention to the possibility of other variables in the eye itself, and it emphasized the importance of hereditary rather than environmental influences. For the first time, myopia was considered to be physiological, depending upon the combination of two factors, corneal refraction and axial length, each of which possessed a normal range of variation.

Steiger did not appreciate that the lens, like the cornea and the axial length, is not a constant but a variable. Its dioptric strength ranges from 52 to 67 in air so that we have three main components combining to produce the total refraction of any eye. If errors of refraction were merely variables on a curve of frequency, it would be possible to plot them on a theoretically derived binomial curve which turns out to be broad and flat, but all curves of frequency of refractive errors in the general population show two important departures from this theoretical one.

1. There are far more cases of emmetropia than would be expected by theoretical calculation. To explain this finding it has been suggested by Tron (1934, 1935) that the different components of the total refraction are not independent variables, and that some process of correlation takes place. From the point of view of heredity this must mean that the various optical components of the eye are not inherited independently from one another.
2. There are more cases of high myopia than would be expected theoretically, with no counterpart on the hypermetropic side. If eyes with temporal crescents and other fundus changes are excluded, or if eyes with myopia of over 6 D are excluded, this excess of high myopes is deleted from the curve of frequency and it becomes similar to the binomial curve. This would suggest that there are two types of myopia—one a physiological variation and the other a pathological process.

Present views

If we regard the lens as a unit, the refractive system of the eye consists of two components, the cornea and the lens, separated by the depth of the anterior

chamber. The relation of the focal plane of this refractive system to the perceptive plane is of decisive importance. If the axial length and the refractive power of one of the components (the cornea) and its distance from the lens are known, we are able to calculate the front vertex power of the lens. The axial length of human eyes can be measured by *x* rays as was shown by Rushton in 1938 or by ultrasonography. The refractive power of the cornea is calculated from keratometer readings and the depth of the anterior chamber can be accurately assessed by the method of Stenström (1946).

What are the results? The power of the cornea and of the lens, as also the value for axial length, show a wide variation (with a normal distribution) in emmetropia, and these same powers and values are observed in myopia up to −4 D. In the emmetropic eye the different powers are co-ordinated with the axial length, but there is no such co-ordination in moderate myopia. The lack of co-ordination is the distinguishing feature of the ametropic eye just as co-ordination is the distinguishing feature of the emmetropic eye. The available evidence suggests that the eye keeps on growing throughout late childhood. The eye becomes myopic, not because the rate of growth of axial length is abnormal—for the axial length of myopic eyes up to 4 D is still within the emmetropic range—but because, during the process of growth, changes in the cornea and the lens have not kept pace with axial elongation.

By contrast, in high refractive errors abnormal components—essentially abnormal axial length—make their appearance. High errors constitute not more than 5 per cent of the refractions seen in the general population but they represent a distinct entity differing from the deviations from emmetropia. They are a problem in pathology as opposed to the low refractive errors which are variations of emmetropia. An anomalous axial length is, however, not the exclusive cause of high refractive errors, for the cornea and lens occasionally come into play. Cornea plana and spherophakia may be the responsible factors for myopia.

The modern views, therefore, of the aetiology of myopia make use of many of the early ideas and findings, but set them within their operative limits: the mechanistic view of the last century, however, with its emphasis on environmental factors is emphatically discarded. Are we about to see a modification of this outlook?

The old problem of nature or nurture is always difficult to solve not least in ophthalmology. It is particularly so where freedom of the individual is paramount. A controlled trial involving the examination of children over their growing period is beyond the possible. Nevertheless this should not inhibit attempts, however imperfect, to answer this question. The paper that follows gives an account of the clinical experience of one individual and one individual only. It is nothing more. Anyone who maintains that it is proof of our ability to arrest myopia in general is either a knave or a fool or both. But there is enough doubt cast on our ideas of environmental influences in the development of myopia to merit further assessment. The environment after all is not a static entity; contact lenses and phenyl epherine were outside the ken of Kepler.

Whether the effects described are temporary or permanent, which types of myopia are influenced thereby, and whether there is a place in clinical ophthalmology for such practices, are all questions raised rather than answered. Lastly it is relevant to bear in mind that a case can be made for the positive advantage of moderate myopia for those living in a developed society such as ours.

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